Third, none of the 11 limbs examined had deep venous reflux extending into the crural veins, and five of 11 limbs had competent popliteal segments. Because in the absence of superficial venous reflux popliteal valve incompetency is pivotal in the development of venous ulceration,[3,4] these five limbs possibly had insignificant femoral vein reflux that did not contribute to the overall calf pump dysfunction. This being the case, one would expect improved venous hemodynamics after saphenous vein surgery. The improved venous hemodynamics reported in this paper reflect this, suggesting that this group of patients who have proximal deep vein reflux behave in a similar manner to those who have normal deep veins.[5] In limbs where deep venous reflux extends across the knee into the crural veins, saphenous surgery confers no hemodynamic benefit,[5] and the authors report two limbs with grade 3 and 4 reflux deteriorating after surgery.

Finally, it is interesting to note the authors' final conclusion in suggesting that saphenous vein ligation is required before deep venous reconstruction. If, as is reported here, such sustainable hemodynamic and symptomatic improvements are possible with saphenous ligation alone, why then do the authors feel it necessary to reconstruct the deep system?

This paper addresses an important and, to date, unclear clinical situation; however, it does not address the role of saphenous surgery in limbs with below-knee or crural deep reflux and unfortunately assesses venous function in a suboptimal manner. As such it fails to clarify the management of patients who have extensive primary deep venous reflux.

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24/41/82085

Reply

To the Editors:

I am pleased to offer the following response to this informed and thoughtful commentary. Limbs with primary, combined, deep and superficial venous incompetence manifested by dermal ulceration were evaluated. After healing the ulcers, superficial and perforating vein components were ablated, and the patients were observed with interval clinical, anatomic, and hemodynamic reassessment.

The concept of venous hypertension is well established as the underlying pathophysiologic mechanism of chronic venous insufficiency. However, the venous filling index (VFI) as measured by the air plethysmograph (APG) is widely accepted as a quantitative measurement of venous reflux.[1-3] Independent investigators who compared the APG measurement of residual volume fraction (RVF) demonstrated a strong correlation ($r = 0.83$) with ambulatory venous pressure (AVP)[2]; the correlation was stronger in limbs without evidence of venous obstruction ($r = 0.86$).[1] Although others were unable to duplicate these results, their data included limbs with prior thrombosis.[4] Notably, none of the limbs in our report had evidence of prior thrombosis. Because AVP measurements—the "gold standard"—do not distinguish deep from superficial incompetence, do not differentiate reflux from obstruction, and do not localize the process anatomically, the addition of imaging is essential when planning management.[1-4] Finally, a critical aspect of the study design involved serial clinical and hemodynamic follow-up; APG provided a reliable, repetitive measurement that was readily accepted by the patients. For these considerations, we did not add invasive AVP measurements to our investigation. The article by Bradbury and Ruckley[5] referenced in the above letter used foot volumetry without other characterization of the deep venous system to assess the long-term follow-up of a similar group of patients. Their findings parallel our report, in that limbs without popliteal reflux had no recurrences over a mean follow-up of 60 months. Notably, however, when an ulcer recurred in their series, it was accompanied by hemodynamic deterioration.

We agree that clinical outcome is the definitive test of the validity of these observations. However, rapidity of ulcer healing or reduction in wound area may be difficult to measure and are subject to other factors besides venous hypertension. Previous work from our institution demonstrated a strong association between active ulceration and reduced calf pump function.[6] Because assessment of the hemodynamic benefits of the procedure was our goal, confounding effects of active ulceration were eliminated by achieving stable healing first. Bradbury and Ruckley[5] also postponed surgical intervention until ulcers were healed. The reported patient group remains intact at a current mean follow-up of 23 months. Ulcerations have not recurred, deep valvular incompetence remains unchanged, and continued improvement in VFI (the mean is now

2.6 ± 1.3 ml/sec) and calf pump function has been sustained.

The authors of the letter note the absence of crural vein reflux. This was discussed in detail during presentation of the data to the American Venous Forum. Although these patients were not selected because of this characteristic, the data support the current importance assigned to the popliteal and crural valve segments. As noted in our discussion, and referenced in their letter,[5] it is likely that popliteal or crural reflux will confer a worse prognosis.

They suggest that "insignificant femoral vein reflux that did not contribute to the overall calf pump dysfunction" may have accounted for some of our findings. On the basis of our data, we question the significance of isolated proximal vein reflux. Six of 11 limbs reported had popliteal valvular reflux. Thus we concur that if these results remain unchanged, one of the conclusions from our data may be that proximal reflux alone may be effectively treated with primary superficial venous ablation alone.

Currently, we disagree with their assertion that superficial and perforator ablation confers no hemodynamic benefit in patients with "deep reflux across the knee" as defined by grade 3 to 4 descending venography. To date, these patients have demonstrated sustained improvement in both hemodynamic and clinical reassessments. Although the magnitude of initial reduction in VFI did not achieve statistical significance, the most recent mean VFI in the grade 3 to 4 group is now 2.3 ± 1.2 ml/sec, which is significantly different ($p < 0.05$) when compared with the preoperative VFI. However, we remain cautious in this recommendation on the basis of the small sample size and duration of follow-up. The alleged "deterioration" in two limbs (page 712) is merely a duplex finding of an additional valve segment with incompetence that was identified during the first postoperative reassessment. The role of deep vein valvular reconstruction is an issue beyond the scope of these letters.

Correction of the superficial venous component in limbs with primary, combined deep and superficial venous insufficiency with ulceration has produced excellent results to date. Outcome in patients who have crural and popliteal valvular incompetence ultimately may prove to be less satisfactory, but our current data do not corroborate this conclusion at this time. As a group, these patients remain as satisfied as any group I have been privileged to manage and will continue to be observed closely.

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Branham's sign is an exaggerated Bezold-Jarisch reflex of arteriovenous fistula

To the Editors:

Nicoladoni-Branham (Branham's) sign, a decrease in pulse and increase in blood pressure that immediately follows the sudden occlusion of an arteriovenous (A-V) fistula, is familiar to all vascular surgeons. Since its description in 1890, it has been considered the standard test of the hemodynamic significance of an A-V fistula.[1] Although the validity of Branham's sign is generally accepted, there have been recent reports of patients who did not exhibit Branham's sign but had seemingly significant A-V fistulae for unclear reasons.

We propose that Branham's sign is an exaggerated Bezold-Jarisch (B-J) reflex. Described in 1867, the B-J reflex causes bradycardia by stimulation of baroreceptors residing in the left ventricle. Certain receptors respond primarily to chemical stimuli (i.e., acetylcholine,[2] 5-hydroxytryptamine[3]), whereas others respond primarily to mechanical effects (volume loading, balloon distension[2]) and some to both. One study showed that the inhibition of nitric oxide (NO) synthesis led to enhancement of the bradycardic reflex.[3] Both Branham's sign and the B-J reflex are abolished by atropine administration,[1,2] and are diminished by the standing position.[2,4]

When a hemodynamically significant A-V fistula is placed in the circulation, cardiac output will rise in an attempt to return systemic blood flow to the baseline level present before the creation of the fistula.[1] Occlusion of the fistula, therefore, momentarily increases systemic blood pressure, reflecting the adjustment period during which the excess cardiac output is forced to pass throughout the higher-resistance peripheral vascular beds rather than the low-resistance fistula. The bradycardic response that follows the occlusion of the fistula occurs within one or two heart beats. This immediate effect has been attributed to be the effect of blood pH on the action of choline esterase.[4] Furthermore, there is now additional evidence that it is initiated by the baroreceptors and is also mediated through